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Blood Lead Concentration after a Shotgun Accident

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In an accidental shooting, a man in his late forties was hit in his left shoulder region by about 60 lead pellets from a shotgun. He had injuries to the vessels, the clavicle, muscles, and nerves, with total paralysis of the left arm due to axonal injury. After several surgical revisions and temporary cover with split skin, reconstructive surgery was carried out 54 days after the accident. The brachial plexus was swollen, but the continuity of the nerve trunks was not broken (no neuroma present). We determined the blood lead (BPb) concentration during a follow-up period of 12 months. The BPb concentration increased considerably during the first months. Although 30 lead pellets were removed during the reconstructive surgery, the BPb concentration continued to rise, and reached a peak of 62 µg/dL (3.0 µmol/L) on day 81. Thereafter it started to decline. Twelve months after the accident, BPb had leveled off at about 30 µg/dL. At that time, muscle and sensory functions had partially recovered. The BPb concentration exceeded 30 µg/dL for 9 months, which may have influenced the recovery rate of nerve function. Subjects with a large number of lead pellets or fragments embedded in the body after shooting accidents should be followed for many years by regular determinations of BPb. To obtain a more stable basis for risk assessment, the BPb concentrations should be corrected for variations in the subject's hemoglobin concentration or erythrocyte volume fraction. **Key words:** blood lead, gunshot wound, health effects, hemoglobin level. *Environ Health Perspect* 110:115–117 (2002). [Online 19 December 2001] <http://ehpnet1.niehs.nih.gov/docs/2002/110p115-117gerhardsson/abstract.html>

During a shooting accident, a man in his late forties was hit at close range by lead pellets from a shotgun. He had injuries to the subclavian and axillary vessels, pectoral muscles, and the lateral part of the clavicle, and clinically he showed a total paralysis of the left arm and hand. Approximately 60 lead pellets were identified by X ray in the left shoulder region. The pellets were 3.5 mm in diameter and weighed 0.25 g, giving a total lead mass of about 15 g.

During emergency surgery the vessel injuries were repaired. During the following weeks, several surgical revisions were undertaken and temporary cover with split skin was performed. A neurophysiologic investigation showed a severe lesion of the brachial plexus, with denervation activity and signs of only a few preserved axons in the musculocutaneous and radial nerves. Fifty-four days after the accident, exploration of the brachial plexus and reconstructive surgery were undertaken. The different parts of the brachial plexus were swollen, but we found no segmental defects in the various nerve trunks that we explored, and we observed no neuroma. There was no reason for reconstructive nerve surgery. The defects over the brachial plexus, remaining parts of the clavicle, and the vessels were

covered with a pedicled latissimus dorsi muscle flap with split skin.

During reconstructive surgery, 30 lead pellets were removed. The remaining pellets had to be left because they were embedded too deeply in the tissues. No blood transfusions were given on admission to the hospital or in connection with the reconstructive surgery, during which the patient lost an estimated 700 mL of blood. Preoperatively, the patient was given intravenous dextran, which was necessary for survival of the pedicled muscle flap used in reconstructive surgery. No chelation therapy was given.

The wounds healed uneventfully. The patient received regular physiotherapy at a rehabilitation unit for one year. Motor nerve function had only partially recovered 12 months after the accident, at which time sensory nerve function was still severely impaired, particularly in the hand. The patient had markedly diminished muscle force and range of motion, especially in the left shoulder. Pronation, wrist flexion, and finger flexion were still absent.

Determination of blood lead concentration. We determined the blood lead (BPb) concentration using flame atomic absorption spectrometry after extraction of

complex-bound lead from fresh blood samples into methyl isobutyl ketone (1). We modified the method slightly by replacing the original complexing agent, ammonium pyrrolidine dithiocarbamate, with diethyldiammonium diethyldithiocarbamate, which gave a more stable lead complex. The method has been in routine use for more than 25 years, and we repeatedly checked the method by internal and external control programs. The detection limit was 1 µg/L. The coefficient of variation for sample duplicates was 8% for the range 9–99 µg/L (mean, 80 µg/L) and 4% for the range 100–930 µg/L (mean, 352 µg/L). The result for a lyophilized reference sample (Seronom whole blood, 905; Nycomed, Oslo, Norway) was 384 ± 19 µg/L (mean ± SD; *n* = 27), against the recommended value of 400 ± 24 µg/L.

Blood lead. We determined BPb concentrations on 25 occasions during a follow-up period of 12 months, beginning on day 23 after the accident (Figure 1). In whole blood, about 99% of lead is in the erythrocytes (2). Changes in the erythrocyte volume fraction (hematocrit) or hemoglobin (BHb) concentration have an immediate influence on the BPb concentration. This effect is shown in Figure 1. For better comparability between BPb concentrations at different sampling occasions, we adjusted all BPb concentrations to the mean BHb concentrations of the patient during the last months of follow-up, 142 g/L (Figure 1). This value is within the reference interval for Swedish males, 131–163 g/L (3).

In the blood sample taken on day 46 (reconstructive surgery took place on day 54), the adjusted BPb concentration was 50 µg/dL (2.4 µmol/L). Although about one-half of the lead pellets were removed during surgery, the BPb concentration continued to rise and

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reached a peak value of 62 $\mu\text{g}/\text{dL}$ (3.0 $\mu\text{mol}/\text{L}$) on day 81. After this, the BPb concentration started to decline; 1 year after the accident, the BPb concentration had leveled off at about 29 $\mu\text{g}/\text{dL}$ (1.4 $\mu\text{mol}/\text{L}$; Figure 1). There was a marked decrease in unadjusted BPb concentrations between day 46 and day 58. The adjusted BPb concentrations did not show a similar decrease.

Discussion

Exposure to inorganic lead may affect the peripheral and central nervous systems, the blood-forming organs, the kidneys, the gastrointestinal tract, the cardiovascular system, and the reproductive organs (4). The accumulation of lead in the endoneurial space in a peripheral nerve trunk may affect the Schwann cells at an early stage. This may be followed by an endoneurial edema, leading to an increased endoneurial fluid pressure (5–7). Such an increased endoneurial fluid pressure may jeopardize the intraneural microcirculation, causing axonal injury (8). Slight effects on the nervous system and kidneys (critical organs) have been reported at BPb concentrations around 30 $\mu\text{g}/\text{dL}$ (4).

In the case of gunshot wounds in which shotguns are fired at close range, a large number of lead pellets may be retained in different parts of the body (9,10). If the number of lead pellets is large enough, dissolved lead from the pellets may cause adverse health effects as time passes (11–14). Symptoms of systemic lead poisoning after shooting incidents may appear after a latency period that varies from a couple of months

to several decades (12,15,16). The impact of retained lead pellets on BPb concentrations depends not only on the number and size of the pellets but also on their location in the body. Solubilization of lead pellets or fragments lodged within or close to joints may increase after a considerable latency time (17). Farrell et al. (16) suggested that disintegration caused by mechanical joint action leads to an increased release of lead into synovial fluid. Chronic inflammation of the joint may follow, leading to arthritis and increased lymphatic and vascular drainage of solubilized lead (16). A prompt removal of intra-articular lead pellets and fragments is therefore recommended (11). This may also diminish the risk of lead-related impairment of nerve function recovery.

The return of motor and sensory function in our patient was dependent on axonal regeneration in preserved endoneurial tubes. During the first 9 months of follow-up, the BPb concentration exceeded the values that reportedly affect the nervous system (BPb > 30 $\mu\text{g}/\text{dL}$) (4). Increased BPb may have impaired the rate of recovery, either by prolonging an endoneurial edema or through direct toxic action on the Schwann cells. Activation and proliferation of these cells are of crucial importance for an optimal nerve regeneration process. Negative effects of lead on recovery rate have not been observed because it is not possible to estimate any “normal” time for recovery after such an unique accident.

The pronounced decrease of the unadjusted BPb concentration immediately after

the operation (Figure 1) can be explained by blood dilution due to blood loss of 700 mL and the preoperative treatment with dextran.

Hemodilution results in a proportional change in BPb concentration. This is due to the very extreme distribution of lead between plasma and blood cells. For some of the BPb concentrations, there were no concomitant BHb concentration determinations. In these cases, we used an interpolated BHb concentration to calculate the adjusted BPb concentration. As is evident from Figure 1, the variation in BPb concentration during the first month after the reconstructive surgery decreased considerably after such an adjustment. For a reliable risk estimation of lead uptake, some kind of standardization of BPb concentrations would be advantageous. In the present case, there was a considerable difference between the highest adjusted BPb concentration (62 $\mu\text{g}/\text{dL}$) compared to the highest unadjusted BPb concentration (50 $\mu\text{g}/\text{dL}$). The current Swedish occupational biological exposure limit for male workers is 52 $\mu\text{g}/\text{dL}$ (2.5 $\mu\text{mol}/\text{L}$), a level at which male workers should be immediately removed from exposure (18).

The significant decrease in BPb concentrations during the 2 months after day 81 was not due to solubilization and excretion in urine and feces of a corresponding fraction of lead (several grams). The transport of such an amount of lead through the circulatory system within 2 months would have been associated with BPb concentrations far above the levels observed. The fact that the amount of lead embedded in the tissues was not reduced significantly during this period means that the decrease in BPb concentration must be regarded as unusually fast. The BPb data for these months indicate a half-life of about 1 week. This is at the low end of the range observed in lead workers after ended exposure, 7–63 days (19). It is probable that the decrease in BPb concentration is caused by incapsulation of the remaining lead pellets in fibrous tissue or by the development of a durable chemical coating around the pellets.

Even if a significant decrease in BPb concentration was displayed during the 12 months of follow-up, BPb concentrations should be determined regularly in the patient for many years. This is especially important in this case because several of the lead pellets are located close to joints, which is a particular risk factor for later mobilization of lead (17).

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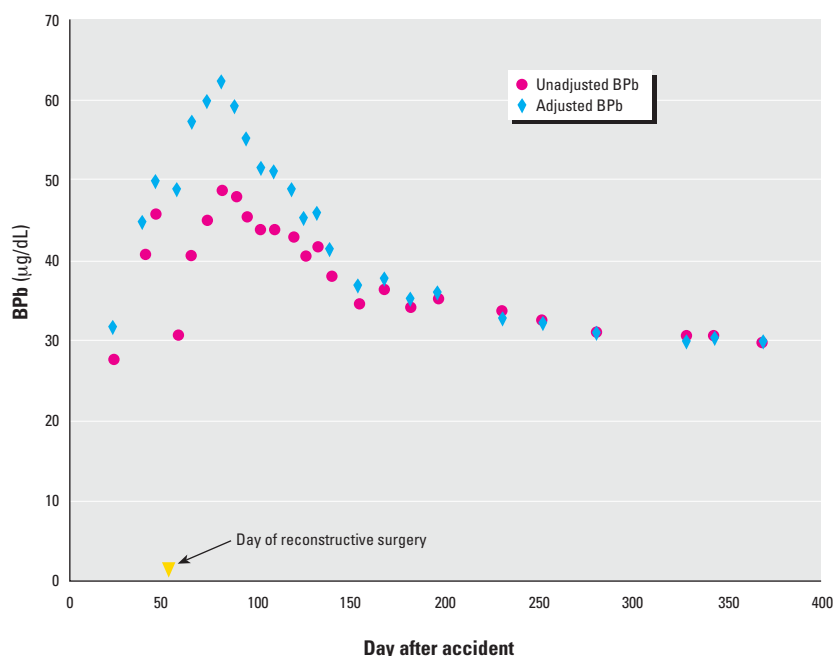


Figure 1. Hemoglobin (Hb)-adjusted (to a standardized Hb concentration of 142 g/L) and unadjusted BPb concentrations ($\mu\text{g}/\text{dL}$) related to the time elapsed after the patient was accidentally shot with a shotgun.

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